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A MONOGRAPH ON DENGUE FEVER.

Thesis for the degree of M. D.

by

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I

A MONOGRAPH on DENGUE FEVER.

Introduction.

Having been through an Epidemic of Dengue Fever whilst stationed with my regiment in Hongkong in the year 1902, I was struck with the very marked contrast of the disease to that described in the text books, and the extraordinary variations which occurred in the cases. I observed also that it was to the European a very much more serious disease than I had been led to think. The fact that all the Asiatic Troops were treated in separate pavilions of the Kowloon Station Hospital gave me the advantage of seeing all the cases admitted to Hospital not in my own wards. Twice a week I had to do duty in the Hongkong Station Hospital for British Troops as orderly medical officer for 18 hours and I therefore saw most of the cases that occurred amongst the British Troops. The European officers were treated in their own quarters and I only transferred one case to the Civil Hospital in Hongkong, on account of pronounced and troublesome nervous symptoms amounting to Melancholia. This case will be referred to later when dealing with the complications. The cases were admitted into the general wards as I was of the opinion

that the disease was non-contagious and not allied to the exanthemata as regards contagion. I had come to this conclusion from observing how only certain individuals from a barrack room would be attacked, and that it never got a real hold in any of the regiments, but appeared only in isolated cases. The men themselves if possible, would not report sick and a few to my knowledge accomplished this when attacked at the end of the week. They do this, as many of them pride themselves on never having reported sick and keeping a clean medical history sheet. Under these circumstances if it had been contagious it could not have failed to spread rapidly. I had the theory that it was a mosquito borne disease from the fact that no hospital assistant, orderly or servant, developed the disease and that whilst the Asiatic troops were comparatively free from the disease, the European troops were far more affected and they again not nearly so badly as the general population. In hospital every case had to sleep with a mosquito net on account of malaria and a rigorous war of extermination of all breeding places of mosquitoes was carried out. The result of the mosquito war which had been in force for some time is very well shown by the fact that out of a European Military Establishment of 1,502 souls including women and children there were only 241 cases.

The European Barracks being situated in the heart of the city of Victoria could not be kept as free from mosquitoes as desired, owing to the proximity of houses and compounds over which Military Control could not be exercised. The Asiatic barracks, and Matsheds on the other hand stood in the middle of their own grounds, and over these the most strict supervision was exercised. Where possible standing water was removed and where impossible once a week crude petrolum was poured over the surface. The results were remarkable. The Sepoys and Native officers of the Hongkong and Singapore Battalion of Royal Garrison Artillery told me repeatedly that never in their experience dating back twenty years had their barracks been so free of mosquitoes. Thus out of a total strength of 2,741 men only 26 cases of Dengue occurred. In the Annual Report on the Vital statistics of Hongkong the following is written. "So badly was the civil community affected that the business of the island was carried on with difficulty, the labour market being disorganised, and the police force crippled".

In the following pages I propose to give an account of the literature on the subject up to the present time and also of my own experiences and observations on the disease as it occurred in the outbreak in Hongkong in 1902.

DEFINITION. Dengue is an acute infectious disease of Tropical and Sub-tropical regions occurring in wide spread epidemics generally from endemic areas. It is characterised by a sudden rise of temperature, an initial erythematous rash, anorexia, pains in the joints and a terminal polymorphous eruption.

GEOGRAPHICAL SURVEY. The earliest extant records of the disease date from the year 1779 when it was described by Dr. Brylton⁽¹⁾ in Batavia Java under the name of "Knockelkoorts" i.e. bone fever, that being the name used by the natives. The same year it occurred in Egypt and was described by the Arab historian Gaberti and was known by the Arabs as "Ahu rokab" i.e. "The father of the knee joint."

In 1780 an epidemic is described by Rush⁽²⁾ in Pennsylvania where it was known as "Break bone fever" or, as one witty lady called it, "Heart break fever," owing to the depression which followed the attack. In the same year Persin a missionary describes an outbreak on the Coromandel Coast.⁽³⁾ (Hirsch).

In 1784 Dr. Cristobel Cubillas describes an outbreak in Cadiz and Seville where it was known as⁽⁴⁾ "La piadosa" i.e. "The pious or benevolent", because no deaths occurred from it. Another outbreak occurred in Cadiz in 1788. No further descriptions of the disease are on record until 1818 when Paredes⁽⁵⁾ describes an outbreak in Lima Peru.

Morice notes an epidemic in Jamaica in 1821 described by Maxwell. The years 1824-5 mark the beginning of the really wide-spread epidemic when Smart draws attention to the Indian epidemic, which beginning on the East Coast at Madras, Pondicherry, Rangoon, Calcutta, spread up the Hooghly and Gangetic plain to Berhampore, Patna, Gehazipore, Benares, Mizapore, and in the same year an extensive epidemic in Gujerat, including its capital Baroda, and also extending to Bombay. Ehrenberg also describes an outbreak at Suez in 1824. In 1826 Waring reports an outbreak in Savannah in Georgia, and in the following years occurred the wide-spread epidemics in the Virgin Islands, St. Thomas,⁽⁸⁾ Santa Cruz, St. Bartholemew, St. Kitts,⁽⁹⁾ Antigua,⁽¹⁰⁾ Guadeloupe, Martinique, Bermudas, Barbadoes, Tobago, Jamaica, Cuba, Curacao, which spread to the mainland of America as far as Charleston,⁽¹¹⁾ New Orleans, and Louisiana in 1831.

⁽¹²⁾
De Brun in 1835 observes the epidemic on the shores of the Red Sea, and shows that it reached Mecca. In 1836 to 1840 it was present in Madras and Calcutta where it was described by Raleigh.⁽³⁾

In 1837 Bermudas (Hirsch)

(12)

"1841 De Brun again mentions the disease in Cairo
and Alexandria.

" 1844 Goodeve in Calcutta and De Brun in Senagal also

" 1847 and 8

" 1845-8 Rey describes ^{an} epidemic in Goree and St. Louis
is in Senegambia.

" 1845-9 Lallemant in Rio de Janeiro and Brazil 1846-9

" 1850 Dickson describes ^{an} outbreak through all the
Southern States of N. America also ^{the} West Indies.

" 1851 Callas in Lima (Smith) Reunion (Dutronlau) and
1852-3 in Peru.

" 1852-3 in Tahiti (Dutronlau)

" 1854 Havana and Curacao, Mobile, Cuba and West Indian
Islands.

" 1853 and 4 a wide-spread epidemic all over India described
by Goodeve.

" 1860-3 Bermudas, Martinique, Senegal, Senegambia,
and Beyrouth. (De Brun.)

" 1865 Senegambia. (Thaly). Canary Islands. (Poggio),
Cadiz (Poggio) Beyrouth.

" 1867 Cadiz (Poggio) and Beyrouth.

" 1868 Port Said (Vauvray) ⁽¹³⁾ and Beyrouth also 1869 and
1870. (Barat) at Reunion in 1869.

" 1870-5 Wide-spread epidemic in the East from Arabia
(14)
and East Africa Tamao, Zanzibar, Mombasa, to

- (15) India to China as far as North Amoy. (16) (Manson.) In 1873 in the Western Hemisphere a more limited epidemic in the Gulf of Mexico, Louisiana and Alabama. 1871-3 Formosa, Java, Celebes and Sumatra.
- 1877 Ismailia (De Brun).
- 1878 Tripoli, Beyruth and Malta.
- 1880 Gairo, Port Said and Alexandria, Ports of Red Sea and Jeddah, East Indian Squadron and Carribbean Coast of North America. (14)
- 1881 Beyruth. Khania to Crete and Syria.
- 1883 Port Said and Beyruth.
- 1885 " " " and Gibraltar, New Caledonia, and Fiji Islands (17) (18) Texas.
- 1886 Beyruth.
- 1887 " Tripoli. (17) Gibraltar, Cyprus, Virginia (Charlottesville).
- 1888 " Aden, Gibraltar, Cyprus. (19)
- 1889 Epidemic over Syria, Asia Minor and Aegian shores of Greece and Turkey and Parts of Black Sea. (20) (21) (22)
- (Cyprus, Rhodes, Athens, Chios, and Islands of Grecian Archipelago, Damascus and Jerusalem, Piraeus, Trapezunt, Varna and Salonica). (17)
- 1890-5 in Senegambia and Hongkong. (23) 1892 Beyruth.
- 1893 E. India Fleet. (24)
- 1895-6 Bombay and in Charleston, Cochin China (Negue) (25)

1897-8 Georgia, Florida and Texas.

(26)

1901 Bangkok

1901 and 2 Hongkong and far East Generally and Austral-
ia especially Brisbane. 1904 Beyrouth.

(26)

(27)

1905 Cuba. (Agramonte).

(28)

(29)

(30)

1905-8 Egypt, Panama. 1907 Philippines.

(31)

1903 Beyrouth.

(32)

1909 Syria

(33)

1910 Syrian and Palestine Coasts.

I have filled in a chart of the World (Appendix IV) shewing the regions affected by the different epidemics with their dates. This shows that the limit of diffusion of the disease may be roughly stated as between the 40th parallels North and South of the Equator. The only time the fever spread North of the 40th parallel was in the outbreak in 1889 when the Southern shores of the Black Sea were infected during a very hot summer. Compare this chart with that of the chart of the reported habitat of the *Culex fatigans*. Appendix V. The striking similarity is very manifest and I have no doubt whatever that if the mosquito was looked for, any differences in similarity at present apparent would speedily be filled in.

SYNONYMS. The name Dengue is now the official nomenclature adopted by the Royal Colleges. Stedman in the Edinburgh Medical and Surgical Journal of 1828 states clearly that the natives of the West Indies mocked their comrades afflicted with the disease, by calling out "Dandy". They did this on account of the characteristic gait adopted by their fellows when they attempted to walk. The pain in the joints of their lower extremities causing them to put their feet down gently, in order to lessen the pain from any sudden jarring. The mincing gait resulting, resembled the careful and affected gait of the jeunesse dorée of those days. The Spaniards in the island called it dunga which means affectation, and so it got corrupted to the present appellation. The names which I notice below are interesting from the fact that they arise from one or other prominent symptom which has chiefly excited the attention of the race which the fever had attacked. The Batavians in 1779 called it Kneckel-keerts i.e. bone fever. (35) Christie states that the natives of Zanzibar in 1822 referred to the disease as Kidingapepo which means the Evil Spirit of Cramp and in 1870 homamagua or leg fever. His contention that the natives in 1870 could remember it being called Denga in 1823 is

untenable. A native with his wish to please is only too ready to profess to remember anything suggested to him. The difficulty in questioning natives whilst endeavouring to arrive at a diagnosis is well known to all medical men in the East, and in these days Western education was not. Therefore the East Coast of Africa can hardly claim the distinction of carrying the name to St. Thomas. But I do not think there can be much doubt that the disease was carried from there as it was called African fever from its appearance after a cargo of slaves had been landed. Gaberti in 1779 mentions the Arab name ^Ahu-rekab i.e. the father of the knee joint, and this name is still known amongst the Arabs for dengue. ^Ahu el thaletta i.e. three days fever is also an Arab name. Vauvray in 1868 in Egypt called it Date fever from its occurring at the time of the date gathering.

The Bunmese knew it as Tek-kive-ana i.e. the tying or drawing together disease or Teehutia, the Hawaiians as Benon i.e. sighs, or Bou-hou i.e. the groaning disease.

The Spaniards have called it La piadosa i.e. the pious or benevolent, because it is practically non-fatal, also ^eCatentura roja i.e. the red fever. Colorado i.e. red, ^aPantomimia and Gauson i.e. burning fever of short duration. In Teneriffe it is known as

Francayo which means a blow with a bar.

The French have called it La fièvre rouge, and in Tripoli père des genoux des massues, i.e. father of beaten knees, also Bouquet from the rash, and the English corrupted it into bucket.

Thaly in Senegal called it the Articular fever of hot countries.

The Brazilians called it Polka fever. In America it was known as Break bone fever, Giraffe or stiff necked fever, broken wing fever.

Among the names given to it in India are Aden fever, as the disease was supposed to have been brought to Bombay by a ship from Aden. Epidemic inflammatory fever of Calcutta. (Mellis). Eruptive epidemic fever of India. The remaining names suggested for the disease are:-Exanthesis Rosalia Arthrodynia (Cock), exanthesis arthrosia, febrilis rheumaticus; scarlatina rheumatica, eruptive articular rheumatism, plantaria, eruptive rheumatic fever.

METEOROLOGY of DENGUE. Dengue is essentially a disease of the Tropics and Sub-tropics. When it passes these limits it does so only at the hottest times of the year and when the humidity of the atmosphere is increased. Directly the weather becomes cooler the epidemic form of the disease rapidly disappears. This is well shown in the epidemics in the Southern States of North America, Egypt, China, and the Mediterranean. It is most extraordinary how the disease clings for preference to coast towns and advances up the estuaries and rivers. ⁽⁴¹⁾ It would appear as if there was some element necessary in the atmospheric conditions for this fact. The only known outbreak which did not confine itself to the Coast line or large rivers was the outbreak in India from 1870 to 1873. In Appendix 1. I have given some figures extracted from the Army Statistics of India ⁽³⁷⁾ (Bryden in 1872) to prove this point. Another point of great interest is that the disease very rarely mounts to high levels. This was most evident in Hongkong. The City of Victoria is built at the foot of a hill known as the Peak. The European Merchants have their residences on it about 1700 feet above sea level and in all the epidemics only two or three cases occurred at that elevation. In Appendix ii I have given the Meteorological readings for the three years during which I was stationed there.

This Appendix shows that judging by these readings there is no meteorological factor to be considered in coming to a conclusion why the epidemic should be bad in 1902 and not so in 1903 and 1904. The only exception to the rule concerning high levels is remarked by (23) De Brun from Beyrouth where the disease spread to villages in the interior from 3,937 ft. to 4,921 ft. above sea level and attacked three quarters of the population.

In Egypt Appendix iii I have extracted from Sandwith's paper the Annual mean temperature and Humidity for Cairo. This shows that the epidemic appears when the Nile begins to rise and continues through the period of the increased humidity. When the cold weather commences it gradually disappears. So long ago as 1827 (39) Ore remarked that the disease on board the East Indiaman "Asia" died out when cooler waters were reached after leaving Bombay. This is the report one gets of epidemics all over the world, that as seen as the cold weather approaches the disease begins to die out and loses its virulence.

(40)
Horton remarks that the meteorological conditions which are ripe for epidemics of malaria are the same for those of dengue.

ETIOLOGY. I think a few remarks are not out of place on the observations and writings of our forefathers in Medical Science who had not the benefit of our highly specialised scientific training and accurate instrumentalia. Their works (I am sorry to say) have, in certain cases been ruthlessly swept aside as unworthy of comment. This doubtless is due to the strenuous endeavour to get at the correct solution of our many problems. The first work I believe extant in the English language viz. Rush of Pennsylvania contains the observation that the epidemic of Break bone fever began in 1780 in the month of August, which was uncommonly warm with a Thermometer standing at 90° F. and mosquitoes extremely numerous. The latter observation he states is a certain sign of an unwholesome atmosphere. How many years have passed! What laborious work has been done! What numerous lives have been lost, to prove that the mosquito net in regions infested by this pest will work as a charm to the health of the users. How difficult too, even at the present day to get communities to take concerted action against the pests. Stedman in Sept. 1827 refers to the fact that the disease appeared in Santa-Cruz when the weather was warm and sultry.

Furlonge in 1830 describes St. John Antigua as one of the dampest towns in the W. Indies being literally built on a general spring and thinks that the fever is due to an atmospheric or meteorological condition and is not communicable. The epidemic occurred when the atmosphere was surcharged with moisture, the days very hot and the nights in comparison cold, this being the ideal condition for driving the mosquitoes into the houses.

Many observers held it was contagious but Wragg of Charleston U.S.A. cites the following:- Its rapid and almost simultaneous diffusion precludes the idea of personal communication. The limited extent of the epidemic, as it prevailed in Charleston, but was not carried into the interior, although the city was daily thronged with people from the country. Its incredibly short period of incubation, the short and uniform career of epidemics in the places where the disease prevailed and its entire and speedy disappearance, this being dependant on climate, season and atmospheric conditions.

Observers all over the world have noticed the fact that infected people coming to an uninfected port start a fresh epidemic and this has been noticed from the earliest times.

Naval Surgeons have drawn attention to the fact that the disease will cling to a ship. This is well shown by ⁽⁴²⁾Smart who reports that the Steam Trooper Dalheusie left Aden in Dec. 1871 with the disease amongst the Lascars. On arrival at Bombay she embarked European Artillery for Cananore. During the voyage from Bombay in Jan. 1872 the European officers of the ship who had been in her from Aden to Bombay, were attacked with the fever. The troops were perfectly healthy on landing but within a day or two were attacked with the disease. From this focus of infection an epidemic spread as far South as Alleypa and Cochin.

Turning to more Modern investigations the work of McLaughlin ⁽¹⁸⁾ first attracts attention by his endeavour to find the causal agents in the epidemic ravaging Texas in 1885. Unfortunately he seems to have formed the preconceived idea that it was of bacterial origin and set out to prove it. This is only too easy in cases where the technique is not perfect and where the whole of one's time cannot be devoted to bacteriological work. He describes a micrococcus which he found in the blood both unstained and stained. He also grew it in an agar culture jelly and in the patients blood drawn into bulbs and sealed. These bulbs were examined, one three weeks after and another three months after the blood was drawn and the same organism

discovered. The statement that the organisms were seen both in the urine and vomited matter, sets the seal on the investigations having been conducted on lines that cannot be accepted as proof.

(3)

Graham of Beyrouth in 1902 made a series of most interesting and conclusive experiments on the mosquito as being the causal agent in the propagation of infection. He noticed that the *Culex fatigans* and *Stegomyia fasciata* were rampant. His first experiments were to prove that if mosquitoes were prevented from biting an infected patient the other members of the household would be immune.

MOSQUITO EXPERIMENTS. 1. A mother nursing her child of eight months old became infected with a severe and typical attack. He at once killed all the mosquitoes in three rooms by means of chlorine and made the rooms mosquito proof. The child did not become infected.

(2). In a family with four children aged 4 to 11. The eldest caught the disease. The above precautions were adopted and although the children were kept together day and night and slept together for 13 days the other children remained free.

(3). A man with a wife and three children. The father became infected. The same precautions were taken and the room kept mosquito-proof for seventeen days and no other case occurred.

The second series of experiments were to prove that the mosquito is the infecting agent.

(1). Four healthy young men were taken from houses free from infection and put under mosquito nets. Then mosquitoes from the nets of infected patients put in.

The 1st case developed a chill after 5 days.

" 2nd " " " " 6 "

" 3rd " " " " 4 "

" 4th " no result.

In the 4th case a new batch of mosquitoes was put into the net with no result. The man gave a history of a severe attack in 1889 so that possibly he was rendered immune.

(2). In case these men might have been infected during the day. He took some presumably infected mosquitoes to a village 3,000 feet up in the hills which was quite free from infection. Here he experimented as before on two young men. One was infected in four days and the other in five. They were kept in mosquito curtains for some days after recovery and all mosquitoes in the house were killed. No other case occurred in the village. These two sets of experiments appear to me to be very conclusive that 1st. the mosquito is the ordinary carrier of infection and 2nd.

that the life cycle of there is one, is extraordinarily short. It points rather to a micro-organism that can multiply with extraordinary rapidity or living in the stomach of the mosquito is injected ^{when} into the insect bites.

BLOOD EXAMINATION. Staining had no result.

When examined fresh an organism with amoeboid movement was seen which in many ways resembled the Plasmodium malaria but most like Piroplasma bigeminum (Borophilus bevis).

Figure 1.     

" II.     

" III.     

" IV.     

Figure 1 shows parasite within twenty-four hours.

" II Later phase of cycle.

" III & IV further advanced and seen pushing its way out.

In a further series of experiments he followed the life history on the body of the mosquito *Culex fatigans*. The blood from the stomach contained the parasite up to the 5th day.

Sporulation Fig. 5.



These spores were found in the cells of the salivary glands in forty-eight hours. Unfortunately this result cannot be accepted. The difficulty in working with fresh blood is due to the refractive changes due to stippling; and is a very real one to the microscopist in Tropical Medicine. The changes occur with such rapidity, that one is deceived into being almost positive that one is dealing with an amoeboid protozoon.

A most interesting experiment however with a mosquito infected 27 days previously is worthy of note. The salivary glands were dissected out and mixed in a warm sterilised normal peptonised solution and injected under the skin of a man who three days later developed a severe attack of dengue.

An unrehearsed experiment also occurred when Mrs Graham in helping her husband with the mosquito cages was bitten on her hand by a fifteen day infected mosquito. Three days later she was attacked by a typical sharp attack of dengue.

(43)
Eberle describes a small dancing and gyrating parasite in the blood plasma but evidently this must have been some contamination.

(44)
Still fails to find any demonstrable protozoan
(29)
Carpenter and Sutton also fail to find any of the bodies described by McLaughlin or Graham.

The last published work on the subject is that
(30)
of Ashburn and Craig of the United States Army Medical Department who were detailed by their Surgeon General to make a study of the etiology in Manila in 1906.

These experiments were carried out in a thoroughly scientific manner. They found no trace of a protozoan or micro-organism either in fresh or stained blood, but noticed the vacuolation which is so suggestive of parasite invasion in the Red blood corpuscles.

Blood Cultures. Rogers citrate method of cultivating the Leishman Donovan bodies was tried and found negative.

Broth Cultures kept for eight weeks were also negative and in four cases contamination occurred but no trace of a protozoan.

They therefore tried intravenous inoculation of unfiltered blood from patients to see if the infecting agent was present in the blood stream. Out of fourteen volunteers eleven were so inoculated, of these seven developed the disease one case doubtful and three absolutely immune.

20 minims of blood was used in each case, taken directly from a case of dengue between the third and fourth day of the disease. The doubtful case is most interesting as half a minim of infected blood was inoculated on Sept. 12th and on the evening of the 14th the patient states that he had a severe headache and pains in the arms and legs. On the 16th only a headache and poor appetite. On the 17th quite well. On the 19th one cc. of filtered blood injected intravenously with no result. On the 25th 20 mms. with no result. This case raises the question as to whether the very minute dose in the first instance caused an immunity by the abortive attack which followed. This also may explain the immunity some persons enjoy from the disease. A minute dose may be injected by one infected mosquito which is sufficient to protect the individual from further infection and the malaise being so trifling as not to be noticed by the patient. This may explain the absolute immunity of the three volunteers who were injected.

Intravenous injection of filtered dengue blood was next tried in two cases. The blood was filtered through a Lilliput filter which was proved by culture experiments to filter off the *Micrococcus Melitensis* and *Spirillum Cholerae*.

10 ccs. of blood taken from one of the above experimentally induced cases was defibrinated and diluted with an equal ^{amount} ~~amount~~ of normal saline and filtered. 50 mms. of the filtrate which contained 20 mms. of filtered blood was inoculated intravenously. In both cases a typical attack of Dengue resulted, in one case beginning 3 days 11 hours and in the other 2 days 12 hours after the injection. This proves that in all probability the organism is ultra-microscopic and is thus akin to yellow fever. It suggests a living agent and not a toxin on account of the incubation period, and the fact that blood from an experimentally induced case of dengue was used.

The Experiments made to prove the transmission of the disease by mosquitoes were not satisfactory as they found great difficulty in handling the mosquitoes in captivity. Of nine volunteers subjected to the infected mosquitoes only one was infected with the disease. As all these men had been subject to the infection before being experimented on, it is quite probable that they had been rendered more or less immune in the manner I have previously suggested.

To the above work I can only add my negative work. I had worked in the Pasteur Institute Kasauli under Colonel Semple and in the Parel Plague Research Laboratory under Mr Haffkine before being ordered to Hongkong; so that I had experience in bacteriological and blood examinations. I invariably examined the blood of every case of fever admitted into my wards. Being interested and intent on trying to discover the causal agent of Dengue I used to go in my spare time to the Hongkong Government Laboratory and work with Dr. Hunter who was then in charge. In all the cases examined both in my own hospital and in the Laboratory we could not discover by any means at our disposal the slightest trace of any micro-organism or protozoon.

That it is a mosquito borne disease probably by the *Culex fatigans* is well brought out by comparing the maps I have added in the Appendices IV & V. I have added a complete description and painting of the mosquito, its eggs and larval stage. Appendix VI.

(45)
Ross in Port Said points out that up to 1905 the place was visited yearly by severe epidemics. In May 1906 a crusade against mosquitoes was begun. Dengue had appeared as usual but rapidly subsided and no case has occurred there since July 1906. In further proof of this being the case a severe epidemic occurred

in Sept. of the same year throughout Egypt. The only places not infected being Port Said and Ismailia where mosquito brigades exist. That the mosquito propagation is more than a theory I do not think can be doubted. From the earliest records we have observers noting that the disease occurred when mosquitoes were most in evidence. Then its seasonal prevalence and occurrence along low lying moist coasts and spreading up rivers. Its rapid diffusion from an infected centre. Its rarity in high altitudes and where mosquitoes are not troublesome. The rapid and almost sudden cessation of an outbreak when the temperature falls. The whole evidence is therefore in favour of a mosquito born disease as it so clearly resembles in its epidemic onset and disappearance yellow fever and malaria. Which diseases are unquestionably mosquito born as proved by the brilliant work of the American Commission and Major Ross.

The question then arises as to how the disease is carried on from one epidemic to another. This is a matter at present of pure speculation. It is not however difficult to imagine that the organism or protozoon may be a natural inhabitant of damp soil and that under favourable climatic or organic conditions be able to infect mosquitoes and so the human race.

Many observers have been of the opinion that the disease is allied to the Exanthemata. This however in my opinion cannot be entertained owing to the fact that the spread of the disease is so sudden and rapid. Every class, race, age and sex suffer equally. That it is not air born or communicated by fomites is well shown by the prophylactic measures taken by Graham in his experiments. He prevented the spread of the disease to the rest of the household by the destruction and prevention of access of the mosquitoes. Ashburn and Craig also proved this to be negative by causing a healthy man to sleep in the beds which had been occupied by three dengue patients and also made him wear their underclothes, the man remaining perfectly well. My previous extracts also show that the writers on the subject were very dubious as to its being contagious or not, some holding that it was, whilst others that it was not.

INCUBATION. This appears to be very variable and dependant on the susceptibility of the individual.

The various writers differ on the subject from one to eight days, but the concensus of opinion is ^{three} about three days. The difficulty in deciding arises from the fact

that ^{it} is impossible to tell when the infection took place and ^{the} absence of prodromal symptoms. In the experimental ^{work} of Graham with infected mosquitoes two cases had an incubation of 3 days

two cases had an incubation of 4 days

" " " " " 5 "

one case " " " 6 "

Ashburn and Craig injecting unfiltered blood from dengue patients.

one case had an incubation of 2 days 18 hours

one " " " " " 2 " 19 "

one " " " " " 3 " " "

one " " "v " " 3 " 18 "

one " " "v " " 4 " 4 "

one " " " " " 7 "

with filtered blood from dengue patients

one case had an incubation of 2 days 12 hours.

one " " " " " 3 " 11 "

Thus the period from experimentally induced cases varies from 2½ to 7 days. It has been reported that a passenger landing in Hongkong from a mail boat was attacked after only twenty-four hours on shore.

IMMUNITY. This is very variable as in some epidemics it is conclusively proved that a second attack may occur; but on the whole one attack in an epidemic generally protects from another. The immunity in some cases undoubtedly holds from one epidemic to another when more than a year has elapsed. Dickinson records in the epidemic in Charleston of 1850 that only those escaped who had suffered from the disease in 1828. In Hongkong I met several men who had the disease in 1901 and had it again in 1902. * There is no doubt a few fortunate individuals escape the disease because they are never bitten by mosquitoes. Charles in the Indian Epidemics noticed that people who had had attacks or had been immune in 1824 were practically the only ones who escaped in subsequent epidemics.

SYMPTOMOLOGY. GENERAL DESCRIPTION of a TYPICAL ATTACK.

The patient is suddenly seized with the disease whilst in perfect health. He has a feeling of chilliness and shivering, and feels generally unwell and very fatigued. So sudden is the attack that very few patients fight against the disease but take at once to bed. The Temperature rises rapidly with a very violent headache and intense pain especially at the back of the eyes and aggravated on movement of the eyeballs. The eyes are injected and watery. The face appears flushed especially over the malar bones, forehead and round the eyes. Its appearance is that of a person vividly blushing and the erythema may spread over all the skin and mucous membranes of the body. This is the initial Eruption and is only an erythema. With this, there is often a diffuse pain over the loins so that the patient cannot lie comfortably. At the same time there is acute pain in one or several joints or in the muscles and is very much worse on movement. Anorexia is present often with actual vomiting especially if the attack comes on after a meal. The Temperature rapidly rises and reaches its maximum in a few hours generally 103° to 104° F. The pulse follows the temperature running from 100 to 120 beats per minute. The skin is hot and dry. At this stage the patient is intensely miserable as any attempt at movement causes an

exacerbation of the intense headache and severe pain in the limbs and loins. The prostration is very marked. The tongue is covered with a moist, white fur. This condition lasts unchanged for usually twenty-four hours, then a slight drop in the temperature with a slight amelioration of the symptoms. Then at the end of the second or third day this stage is terminated by crisis with diaphoresis, diuresis and perhaps epistaxis.

All the urgent symptoms disappear and the patient becomes quite comfortable. This is the Interval when the temperature becomes normal and the patient may want to get up and go back to his work. All he feels, is a slight twinge in his legs or loins and slight giddiness. The tongue cleans and the appetite and feeling of bien-être returns. This lasts until the fourth or fifth day from the beginning of the attack. Then a rise of temperature occurs usually not more than 102° and only lasting a few hours. With the recurrence of the fever an eruption of a rubeolar character appears, known as the Terminal rash. It first makes its appearance on the palms and backs of the hands and from there rapidly spreads to the arms, trunk and legs. The eruption continues for a variable time generally one to two days and then disappearing followed by an imperfect furfuraceous desquamation. The patient in an ordinary attack rapidly picks up strength and his appetite returns, and after a few days debility is restored to

perfect health with perhaps now and again a twinge in the back or joints to remind him of his attack.

The above general description corresponds on the whole with the epidemic that I have observed. The initial erythema was by no means always present and when it was, the patients often complained of a feeling of tightness in the skin and there was a certain amount of fulness of the skin to be made out. The terminal rash also was very often absent and so was the rise of temperature, the patients convalescing straight away from the third day.

MODE OF ONSET. In nearly all accounts the sudden onset has been noticed. At times with definite rigors but more often the feeling of chilliness which has very often been present all through the attack.

(8)
Stedman observes that his patients had a feeling of intense cold through all the stages. The joint pains of a sharp shooting character are usually in the ankles, wrists, and knees and their onset is sudden, corresponding to the feeling of chilliness. A common onset is for the patient to wake out of sleep with great pain in the head, joints and loins; or to be attacked soon after he gets up and before he has finished dressing. In certain cases there are definite prodromal symptoms the patient having a feeling of languor, anorexia, and

a dirty tongue the day before he is definitely attacked. In many cases if the temperature was taken it would be found to be over 100°F. ⁽⁴⁶⁾ De Brun has noticed cases in which the temperature did not rise in the beginning of the attack. The initial erythema is a sign that must not be trusted. At times it is very marked at the onset, at other times it appears from twelve to fifteen hours later and very often is not present at all. The eyes are usually painful on movement from the first and the headache is frontal, deep seated and throbbing. The conjunctiva is injected and watery and the description of ferret eyes is very correct. The attack may begin with vomiting and nausea and uneasy sensations in the epigastrium but in the majority of cases only a distaste for food. ⁽³⁾ Cabillas states that patients were taken at the beginning with vomiting and abdominal pains until the fever was established, others by a strong watery diarrhoea, others by a state of dilation and inertia of the stomach accompanied with great feebleness, distaste for all food and a particular repugnance for the drinks. Together with these more or less acute symptoms there is an extraordinary degree of mental and bodily lassitude. The patient feeling tired and weary beyond anything he has ever experienced before.

FEVER. This I found to be most irregular in its behaviour. As an almost invariable rule the temperature rapidly attained its maximum; that is within four or five hours of the onset of the symptoms. From this point it was impossible to foretell how long the disease would last. To my great surprise I have seen sharp attacks suddenly end by crisis after twenty-four hours. These were accompanied by profuse diaphoresis and diuresis and the complete disappearance of all the unpleasant symptoms, and no return of the fever as far as I could ascertain. In other cases there would be only a slight remission not more than one degree on the second day and the temperature usually dropped on the evening of the third day. In certain cases it would not drop until the fourth or fifth day; and in these cases the terminal rash usually appeared as the diaphoresis came on. With the fall of the temperature the acute symptoms disappeared as if by magic. The only active sequelae being pain in the joints and muscles of the extremities on sudden movement. In very few of the cases did I see the typical return of the fever on the 4th or 5th day with a rash that was apparent. In my hospital cases my Hospital Assistants most zealously watched for it, as in my first cases I had expressed an opinion that they must have missed it as so transient. In my private cases the difficulty of detecting a fleeting rise of temperature is not surprising.

Turning to the literature on the subject Rush in Pennsylvania in 1780 observes that the solution of the fever usually took place on the third or fourth day and does not mention a second rise. ⁽¹¹⁾ Dickson in Charleston on the other hand in 1828 describes the initial fever as lasting on an average thirty-six hours and on the sixth day a second paroxysm. ⁽⁸⁾ Stedman and the writers on the epidemic in the West Indies in 1828 all observe that the first attack usually lasted thirty-six hours and was followed on the third or fourth day by a slight secondary fever. ⁽¹⁵⁾ Charles in the Indian Epidemics notes how rarely the secondary fever occurred and when it did so, was never over 100°F. Sandwith in the numerous Egyptian epidemics notes the temperature as rising to 104°F. and dropping to normal on the third day and in many cases a rise on the fourth day which may last three days. The Greek Medical Committee describe the initial fever as rising to 104° to 105.8°F. and lasting from 24 to 36 hours, then after a remission of two to three days another slighter rise. Negue in Saigon noted an initial temperature of 104° to ^{105.8°} ~~105°~~ F. lasting 48 hours, then a return after one to two days' interval lasting 24-36 hours. Four of his cases terminated fatally and in them the temperature rose to 107.6°F. before death. ⁽⁴⁷⁾ Shoo in Penang and Pridmore ⁽⁴⁸⁾ in Burmah in 1902 both noted that the temperature rose to 104° or 105°F. and then from its maximum gradually

fell to normal on the third day, and that any further rise of temperature was very rare, although the secondary rash was very evident. (49) Jones in the Philippine epidemic of 1907 has made the most interesting observations showing how in the same epidemic the temperature charts differ and gives three varieties.

(1). Temperature rises to 103°F . for 36 hours and then drops by crisis to normal. It rises again to not more than 100°F . and is irregular between normal and 100°F . for two or four more days.

(2). A temperature rising to 103°F . or 104°F . and remaining there for twelve hours and a gradual fall through two or three days to normal and no secondary rise.

(3) The temperature as before rising to 103°F . or 104°F . and after 24 hours dropping to 99°F . The same evening rising again to 103°F . or higher to drop again a little in the morning and to remain irregular for five to seven days before returning to normal.

THE RASHES. The Classical accounts of the disease all describe two rashes. The first or initial rash is a true erythema that may be confined to the face or spread all over the body. In some cases it is accompanied with a feeling of tightness of the skin and a visible and palpable oedematous condition but this latter accompaniment is not common. The rash may be present from the very first or may not develop for twelve to fifteen hours after the onset of the fever. Again the rash may not appear at all. In certain cases it is accompanied with a hyperaesthetic condition of the skin. De Brun noticed that if the weather was very hot this rash was not marked whilst on the other hand when the weather was comparatively cool it was very well marked. This may point to the fact that when the skin was well supplied with blood it was able to get rid of toxins without causing a hyperaemia. In the truly tropical outbreaks this initial eruption is not such a constant feature of the disease except in the Calcutta outbreak of 1824 when the skin was described as being hot and dry and covered with a uniform blush.

The Terminal rash in the majority of epidemics is well marked but again it may be so trifling on the extensor surfaces of the legs and arms that it may be missed. It is also at times completely absent. This rash begins as a rule on the third day but not definitely confined to that day. A greater latitude must

be given to the day on which it makes its appearance I would say from the beginning of the third to the sixth day. The rash usually begins on the palms of the hands and soles of the feet and has no definite type of its own. ⁽⁵⁷⁾ Castellani's description is excellent. The rash is of two types (1) A measly eruption of small circular dark red papules which almost entirely disappear on pressure or (2) a scarlatiniform eruption of close set bright red points which may coalesce to form large red patches. Eruptions intermediate in appearance between these two types may be present.

In the accounts of the epidemics it is frequently noted that there is a definite swelling of the hands and feet together with numbness and an itching burning sensation of the skin. The rash is present for two to eight days and in the majority of cases is followed by a fine furfuraceous desquamation, which may continue from one to two days to two or three weeks. In rare instances an intense pruritis may be present. The rash is never petechial but always disappears on pressure. It usually disappears in the order in which it appears namely first on the wrists, hands and feet then on neck, legs, and trunk. Usually with the appearance of the rash there is a rise of temperature, especially in these cases where the rash makes its appearance after the temperature has fallen to normal.

(23)

De Brun notes in the Syrian epidemic in 1892 that he has seen cases of Dengue in which the only symptom has been the eruption, also where all the symptoms of the disease were typical together with rash all over the body but no temperature. This epidemic was marked by the eruption appearing in every case of the disease. There appears to be no relation between the severity of the disease and the rash, but entirely due to the idiosyncrasy of the toxine in certain epidemics and to that of the individual in others, combined with the mean temperature of the atmosphere.

JOINT AND MUSCLE SYMPTOMS. Pain is a constant and early symptom in all epidemics. The severity is not fortunately always the same, as in bad cases it can be described as though a red hot iron was being thrust into the joints. The curious point is, that the pain cannot be definitely localised by pressure, and that it is more peri-articular than inter-articular. Very frequently it is not in the joint at all but entirely muscular, and very liable to attack the lumbar muscles. The pain comes on suddenly and affects especially the wrists, ankles, small joints of the fingers and toes and knees. Swelling and tenderness around the joints may be present but in the majority of cases is not apparent. The pain is immediately relieved on defervescence of the fever. In many cases it disappears altogether. In others although the pain is relieved it is present for several days after the patient has resumed his ordinary occupations. It is then worse after rest but on using his joints freely is ameliorated. In the majority of cases where the pains persist they are present in the joints of the fingers and toes. In these cases very frequently slight swelling and tenderness round the joints is apparent. Ladies have frequently told me they could not get their ordinary gloves on. I have known cases where patients four

weeks after an attack has been recovered from have been suddenly incapacitated from an attack of acute pain in the ankle. If it had not been for an entire absence of constitutional and local symptoms one would have feared the development of some grave condition. These attacks ^{are} particularly liable to come on if the patient catches a chill. The acute pain during the fever is, I think, far more muscular than arthritic and is only referred to the joints. My reason ^{for this} is, that on handling a limb and making gentle movement of the joints no aggravation of the pain is present. On asking the patient to resist the movement he at once complains of very severe pain about the joint, but has great difficulty in localising it exactly.

No true atrophy of the muscles follows but only slight wasting from disuse. There is no special tenderness over the nerve trunks.

CIRCULATORY SYSTEM. This system is not affected to any great extent. The only observations that I can find are that in some epidemics there is a tendency to giddiness; many observers have noticed that the pulse rate is slow in comparison with the height of the temperature. Sandwith observes that the pulse may be quiet and soft beating from 84 to 90 to the minute although the temperature is 104° F. As a rule the pulse taken in the morning is never over 102 beats to the minute whilst in the evening not over 108. In some epidemics the pulse is as rapid as 120 at the height of the fever but no higher pulse rate has ever been recorded. A very common phenomenon is for the pulse rate to be diminished to 50 or 60 after the temperature has fallen by crisis. A troublesome palpitation may occur during convalescence but it is not accompanied or followed by any organic disease of the heart.

HAEMORRAGE. In very many epidemics in all parts of the world epistaxis has been a well marked feature of the disease. This usually occurs at the crisis and is at once followed by great relief, the pains in the head and behind the eyes being relieved at once. The bleeding may be profuse but is never dangerous and only in very exceptional cases is it necessary to plug the nares.

In some epidemics especially those described in 1880 by Eugene Foster of Augusta and D Aquin of New York there is a marked tendency to haemorrhages from the nose, gums, bowels, stomach, lungs and uterus. In this epidemic the convalescence was very slow and wearisome. Probably due to the debility following the loss of blood. In the Smyrna epidemic of 1889 besides the haemorrhages noticed above haematuria was present.

BLOOD. The observations on the blood are at present very inadequate; due no doubt, to the fact that 1st the epidemics are generally so severe that the medical man has not the time to conduct the accurate observations that are necessary, and 2nd that no demonstrable parasite has ever been discovered, and much time has been spent in trying to discover one at the expense of making blood counts. Every one agrees that anaemia is not present. The following are the records of such work.

(27)

The Queensland Branch of the British Medical Association instituted an enquiry into the Epidemic in Brisbane in 1905 and no causal agent could be discovered. The findings of the Differential blood counts were as given below.

Day of Disease.	Lymphocytes.	Large Mononuclears.	Poly-morphonuclears.	Transitional	Eosinophile.	Amebic.
1 st day	3.3	7.2	81.3	-	-	8
1 st "	29.6	6.0	61.0	3	-	-
2 nd "	29.0	8.6	61.0	-	-	-
2 nd "	17.0	25.0	57.0	-	1	-
2 nd "	8.0	30.0	60.0	-	2	-
3 rd "	23.0	20.0	54.0	-	8	-
3 rd "	26.0	17.0	57.0	-	-	-
4 th "	16.7	20.0	48.3	2.9	0.5	11.6
4 th "	18.4	19.6	58.6	-	0.4	2.2

The above observations show a decrease from normal of the Polymerphonuclear leucocytes and an increase of Large Mononuclears and an extraordinary diversity of observations on the Lymphocytes. From the above records nothing definite can be made out except the increase in the mononuclears.

(32)

Balfour gives the following as the result of his observations in Egypt in 1906.

Day of Disease.	Lymphocytes	Large Mononuclears	Poly-morphonuclears	Transitional	Eosinophiles	Basophiles	Myelocytes
1 st day.	5.25	8.0	84.25	0.5	1.75	0.25	—
2 nd "	25.0	12.0	60.0	1.75	1.25	—	—
2 nd "	48.5	14.5	33.5	1.0	2.5	—	—
3 rd "	32.0	7.5	57.25	1.75	1.5	0.25	—
3 rd "	53.25	10.25	35.25	0.25	0.75	0.25	—
Note Noted	42.5	27.0	27.25	1.25	1.75	0.25	—
" "	52.25	5.0	27.75	0.75	12.5	0.5	1.25
" "	38.5	12.75	43.0	3.75	2.0	—	—
" "	22.25	26.0	44.25	1.25	6.25	—	—

Here the Polynuclear leucocytes are undoubtedly diminished in number whilst the large mononuclears are increased and the lymphocytes very much so indeed. No diagnosis as to the day of the disease from the blood count could be formed from the above data.

(30)

Carpenter and Sutton made two series of experiments in Panama on the Differential leucocytic count at different stages of the disease.

Stage of Disease.	Lymphocytes	Large Mononuclears	Poly-nuclears	Transitional	Eosinophils	Mast Cells
<u>1st Experiment.</u>						
On Admission	23.0	7.9	32.2	36.6	—	0.3
1st drop of Temp.	46.8	5.4	41.0	6.7	—	—
Quiescent Stage	47.0	6.3	40.6	5.8	0.3	—
2nd rise of Temp.	33.6	7.6	50.8	5.2	4.0	—
2nd drop of Temp.	25.1	5.9	52.5	5.4	11.0	—
Convalescence	30.3	5.3	50.0	4.2	9.7	—
<u>2nd Experiment.</u>						
On Admission	36.3	3.8	52.4	7.1	0.4	—
1st drop of Temp.	34.3	3.9	54.0	6.4	0.3	0.1
2nd rise of Temp.	17.8	6.9	69.4	5.6	—	0.3
2nd drop of Temp.	38.2	6.9	52.1	2.4	0.3	—
Convalescence	45.9	6.3	33.1	9.3	3.0	—

From the above tables we have a very definite decrease in the number of polynuclears and a rise in the Large mononuclears; if we include the Transitional with the large mononuclears it becomes very marked. The Lymphocytes in all but three observations were very much increased. The Eosinophil cells seem to become more numerous as the disease progresses.

Ashburn and Craig in Manila in 1906 have made the most extensive observations on the blood in Dengue. Their results bear out the work of the previous writers. They found as follows.

Haemoglobin and colour index normal.

RED BLOOD CORPUSCLES. No anaemia, and never found the red cells under 4,500,000. The size of the corpuscles is unchanged. Poikilocytosis is not common but may occur at the height of the fever. Vacuolation is very common both in fresh and unstained blood. It is most suggestive of parasitic invasion and must be carefully guarded against in making observations. It is not uncommon to observe cocci or bacilli either in the blood plasma or attached to the Red Blood corpuscles but these are due to contamination.

LEUCOCYTES. Marked leucopenia. The lowest number recorded was 1,200 per cubic millimetre and the highest 4,860. The average was 3,800 per cmm. This character is progressive and most marked on the 5th or the 6th day. No morphological change occurs in the leucocytes. There is a decrease in the polymorphonuclear cells and an increase in the small lymphocytes.

In the blood taken from a case of experimental intravenous inoculation of filtered blood in which the differential count was carefully carried out. The results were.

	Polymorph- onuclears.	Small lym- phocytes.	Large lym- phocytes.	Eosinoph- iles.
1st day of Disease	50.0%	41.0%	7.5%	1.5%
3rd day	52.0%	36.0%	8.0%	4.0%
6th day	48.0%	14.0%	32.0%	6.0%

Vedder who carried out hundreds of counts on their behalf found the Polymorphonuclears greatly decreased, the small lymphocytes greatly increased and the large lymphocytes moderately increased during the later days of the illness.

BLOOD PLATES. No change either in number or appearance.

BLOOD PLASMA. No organism was found of any etiological significance and only in one or two instances contaminations were present.

ALIMENTARY CANAL. This is affected only as part of the general toxaemia and no definite symptoms are characteristic of the disease. The tongue is coated with a moist white fur which in severe cases becomes dry and yellow. Very frequently there is a most disagreeable taste in the mouth. Nausea or actual vomiting with abdominal pain may occur at the commencement of the attack. Great distaste for all food and what is very curious is the distaste for liquids which is practically peculiar to this fever. In certain epidemics diarrhoea and haemorrhage from the bowels, but the latter is rare. Haematemesis has also been noted. Constipation is the general rule. A most painful condition of the tongue and mucous membrane of the buccal cavity has been described in certain epidemics from the days of Rush. They have been described as Aphthous sores. They consist of isolated greyish vesicles one sixteenth of an inch in diameter, each surrounded with an area of congestion, are very painful and usually appearing on the second or third day of the fever. These small vesicles burst leaving a small superficial ulcer which soon clears up when the temperature falls to normal. A common condition is a sore throat, which is marked by a dry congested appearance of the mucous membrane.

The liver and spleen are not enlarged nor is there any tenderness over the abdominal viscera.

RESPIRATORY SYSTEM. This system is practically unaffected. The respiratory rate varies with the temperature being raised in proportion to the increase in the temperature. In certain epidemics a slight nasal catarrh has been observed. This absence of any respiratory trouble is useful in diagnosis from Influenza. De Brun has noticed that owing to very severe pain in the chest the patient may be prevented from lying down. The pain necessitating very frequent and short respirations. Charles of Calcutta describes certain fatal cases where the lungs became oedematous and the patient became comatose.

NERVOUS SYSTEM. Is affected secondarily to a marked toxæmia in severe cases. The symptoms are a great irritability of temper and extreme restlessness. Insomnia is frequently very troublesome and at night there may be a certain amount of delirium especially with a high temperature. In children delirium and convulsions are common especially when the temperature reaches 104°F. or over. In some cases I have remarked an extreme nervousness on the part of the patient and a fixed belief that he is really suffering from a very serious illness with great alarm about his condition. This is out of all proportion to the severity of the attack. It was observed in the Hongkong epi-

emic of 1895 that Delirium going on to coma occasionally occurred and also maniacal and suicidal tendencies were present in very severe attacks. A peculiar hyperaesthetic condition of the skin has been noticed. Sandwith has observed in Egypt that in convalescence from severe cases, the patient may suffer from impairment of memory, sleeplessness and even mild delirium. In other cases trying ^{and} debilitating neuralgias are apt to occur and partial paralysis has been noted in one or more limbs. As regards Special senses the eye is the only one ever affected. In this there is a suffusion of the conjunctiva almost amounting to conjunctivitis with an increased secretion from the lachrymal gland. The eye muscles seem to be especially liable to be implicated and to be excessively painful on movement.

RENAL SYSTEM. The kidneys are never affected and albumen is practically never present in the urine as a result of dengue. The urine is high coloured, never suppressed. Painful micturition and haematuria have been noticed but excessively rare.

GENERATIVE SYSTEM. Orchitis has been present in many epidemics but is transient and uncommon.

Metrorrhagia is very common in certain epidemics and there seems to be no doubt that abortion may occur as a result of the disease.

HAEMOPOIETIC SYSTEM. The spleen is never enlarged. The lymphatic glands have been described as being enlarged and tender in many epidemics in all quarters of the globe. In the West Indian and American epidemics in 1828 it was noticed that the Cervical, Axillary and Inguinal glands were swollen and painful; coincident with the outbreak of the Terminal rash. In the American epidemic of 1880 besides the enlargement and painful condition of the glands the unique observation was made that these glands remained in this condition for several months. The glands are by no means always enlarged and in only a percentage of cases are they palpable or painful. The highest percentage of cases in which the glands were enlarged was in the epidemic in Burmah in 1902 when 75% of all the cases had enlarged glands. These were the Superficial cervical, axillary, inguinal and supra-condylar glands. When future epidemics occur with enlarged glands, I would think, that there would be a greater chance of finding the causal agent of the disease in one of these glands. In the epidemic in Hongkong in 1902 I saw no cases with enlarged glands.

VARIABILITY OF EPIDEMIC TYPE. In this fever

no very definite type of disease is universal. In the same district it very frequently happens that the following year ~~one~~ of the most characteristic symptoms of the former outbreak is completely wanting or definitely altered in character. (28) De Brun of Beyreuth who has had the opportunity of seeing many epidemics writes that no two epidemics are exactly the same. "One epidemic will be characterised by skin eruptions in all cases whilst in others no eruption is visible from start to finish. The temperature differs very much, the majority of epidemics being characterised by high fever but again the fever may be so mild as to be absolutely of no account. In some epidemics great pains and headaches whilst in others a condition resembling the end of a typhus."

From all writings that I have read on the different epidemics I have gathered the following symptoms which are common to all. The sudden onset of the disease with a distinct nervous depression and feeling of acute illness, suffusion of the conjunctivae with pain in or behind the eyeballs. Severe headache generally frontal. Sharp shooting pains in the back and extremities. A rise of temperature in the majority of cases to above 102° F. A complete distaste to all food if not actual nausea. The actual illness is of

short duration the patient being convalescent after a week or less. These are invariable and I will now ~~am~~ shortly discuss the main features of what may be the different courses of the disease. The course of the Temperature may be divided into three groups. (1) A high initial temperature of 103°F. or 104°F. which is maintained from 24 to 48 hours and then drops by crisis to normal and no further rise takes place.

(2). The same high temperature but instead of dropping by crisis it gradually and steadily falls to normal through two or three days.

(3). A high temperature from 102° to 105°F. that drops by crisis after 24 hours to normal or 99°F. and remains so for 36-48 hours and then suddenly runs up again to 101° or 102°F. and remains elevated for two or three days.

The rashes are very variable the erythema being absent in many epidemics, if it does occur, it is always within the first 15 hours of the initial rise of Temperature.

The terminal rash when present in the cases of fever in the 1st and 2nd groups may appear when the temperature reaches normal or may appear any time within eight days after the temperature has reached normal. In the 3rd group it almost invariably accompanies the second rise of Temperature. The rash generally fades in two days but may last much longer and may or may not be irritable. Desquamation usually follows.

The joint pains may or may not be accompanied by swelling and are variable in their intensity. The after effects ^{may} in some cases, especially if the patient is exposed to chill, be very severe and simulate the pain of an acute rheumatic condition.

COMPLICATIONS AND SEQUELAE. These fortunately are distinctly rare and as a rule the patient in a few days is in a fair way to recover his normal health. In the epidemics in the near East bronchitis, Catarrh pneumonia, vomiting, diarrhoea, intestinal, bronchial, uterine and vesical haemorrhages are described by the (20) Greek Medical Committee in 1889. Two cases of myelitis were also put down as the result of Dengue. In Egypt the sequelae may be a mental and physical prostration with persistent and annoying insomnia, over which narcotics have little control, and anaemia may occur. Great nervousness with trying and debilitating neuralgia, severe diarrhoea, furuncles, abscesses and partial paralysis of one or more limbs.

Hyperpyrexia is rare and pleurisy, pericarditis, endocarditis and meningitis sometimes complicate the disease and alter its characteristics.

The lymphatics ^{gland}, especially the superficial cervical, axillary and inguinal are sometimes enlarged and painful.

Two cases I had charge of in Hengkong are very interesting on account of the complications. The first was in a Colonel of the Indian Army aged 48. He passed through a typical attack of Dengue and his temperature was normal on the fourth day, never having been above 103°F. He had been very depressed from the first but not more than might be expected in a healthy active man suddenly confined to bed with a painful illness. On careful examination I could not discover the slightest abnormality and he only complained of indefinite pain in the joints of his feet, ankles, and legs. He however informed me that he was very ill and certain he was going to die, which he did not wish to do on account of his being a family man. All persuasion, explanation and tonic treatment being of no avail I persuaded him to enter the Civil Hospital where he could be carefully nursed. On admission there the Medical Officer confirmed my opinion as to no organic disease and it was not until after a month that he was able to return to duty. His family and past history being excellent there was no cause ascertainable for this condition except as the result of some functional affection of the higher centres in the brain.

The second case was in a Subaltern aged 22 who had a very mild attack and resumed duty a week after with a warning to take things easily and not to

overtire himself. For ten days he was perfectly well and he then played for his Company in a regimental check-
 ey cup tie. That evening at Mess he complained of being very tired and having severe pain in his legs, and said he had played a very much harder game than he intended. The following morning I told him not to go on to Parade and I examined him in bed and found he was suffering from a multiple neuritis of both lower extremities. The muscles were extremely painful when grasped and there was pain on pressure over the nerves. Movement of the limbs was painful and difficult. Constitutional symptoms were absent except he said he felt very weak and slack. Fortunately his orderly was a skilful masseur and with that and strychnia internally and later the faradic current he very rapidly improved. Within a month he was able to get up. The left leg was then not painful and the muscles only slightly wasted from disuse. The right leg was more wasted and slightly painful but no paralysis. He was extremely anxious to go on leave to Japan with a friend of his who promised to look after him. After consultation we let him go thinking the week's voyage and cooler climate would be beneficial. He improved rapidly and forgot our warning, with the result that the neuritis came back in the right leg and settled down in the sciatic nerve. He was treated in Yokohama and on expiration of his three months leave returned to Hong-

--keng much worse than when he went away. There was considerable wasting of the hamstrings and leg muscles but no electrical changes and no sensory changes, and no pain. The reflexes were slightly exaggerated. I would have been inclined to think if it had not been for the wasting that it was an hysterical manifestation as his father and grandfather both died after a stroke and he was then very nervous. A Medical Board invalided him home and could come to no definite conclusion as to the lesion. In London he placed himself under a nerve specialist who besides electrical treatment encouraged him to use his leg as much as possible. He became rapidly worse and found great difficulty in walking. On consulting Sir Victor Horsely who thought it was probably meningial as a slight scoliosis was then present; he ^{was} ordered rest, massage, and a liberal diet. Under this he rapidly improved, so much so, that before the year was finished he was practically well and thoroughly able to do his work in the Accounts Department to which he was transferred. When I saw him in India four years later I found the right leg definitely smaller than the left leg but no signs of any paralysis or sensory changes. I may be wrong in the conclusion that it was the result of Dengue but in the absence of any signs or history of venereal trouble etc. it is very difficult to conceive of a reason in a previously thoroughly healthy athletic well developed six foot subaltern.

MORTALITY & MORBID ANATOMY. It is~~s~~ most exceptional for the disease to be followed by a fatal result except in young children and chronic or aged invalids. Children may succumb if the temperature is allowed to rise above 104°F. Delirium and convulsions then determining the fatal issue.

In the Calcutta epidemic described by Charles he saw rare cases which were fatal. In these the lungs became oedematous, the patient drowsy and cyanotic and passed into a comatose condition with hyperpyrexia and death.

Paine in Texas 1885 observed a localisation of inflammation on the serous membranes especially pleura and peritoneum.

Hirsch mentions ~~serious~~ infiltrations in the vicinity of the joints, and reddening of the crucial ligaments of the knee joints.

(25)
Nogue in ~~Sa~~ in 1897 gives the results of four post-mortems he performed on cases that died from the disease. The temperature in the fatal cases rose to 107.6°F. Congestive phenomena increased and the patients fell into absolute coma. Face anaemic, injected and swollen. The eyes projecting and watery, the pupils almost insensitive. A profound stupor is painted on the countenance. The limbs are flaccid.

Sensibility is abolished. Respiration is laborious, distressful, stertorous, and the lungs full of rœchi.

The Autopsy showed in every case some phenomenon of congestion, of two organs in particular the lungs and the brain. On the surface of the lungs an effusion of blackish blood which was present also in the pulmonary tissue. The lungs were of a blackish colour on section. In the cranial cavity the meningeal vessels were dilated and injected so that their ramifications were most marked. The meninges were adherent to the brain. There was nearly always present a liquid sero-purulent infiltration in the meshes of the pia mater. The cavities of the heart always contained a well marked quantity of blackish fluid blood.

The liver, spleen and kidneys also showed some phenomena of congestion but did not offer anything particularly worthy of special notice.

Externally on the dependant parts of the body well marked ecchymosis was very apparent.

Three of the four cases were in patients of strong constitution; the fourth was adipose but well preserved and showed little post-mortem rigidity.

DIFFERENTIAL DIAGNOSIS. When an epidemic is raging the diagnosis is easy but the first few cases often present great difficulty especially in the Tropics. Now that the mosquito has been proved to be the chief element in the spread of the disease an early diagnosis is of especial benefit, to prevent the spread of infection. Dengue fever must be diagnosed from malaria, scarlet fever, measles, small pox, yellow fever, rheumatic fever, influenza and seven day fever.

MALARIA. The recognition of the *Plasmodium* in the blood is absolutely diagnostic of malaria. The leucocytic differential count and absolute count in malaria will show that the large mononuclear leucocytes are increased in number, may be pigmented and that there is a leucopenia; whilst in dengue it is the small lymphocytes which are increased in number. The general onset with acute shooting pains and erythema are characteristic of dengue. The stages of a typical attack of Malaria, the temperature chart, action of quinine etc. are usually quite sufficient to distinguish the two diseases.

SCARLET FEVER. In the Tropics the disease is excessively rare. The presence of an oedematous ulcerated sore throat, with enlarged brawny cervical glands, strawberry tongue and absence of muscular and joint pains make the diagnosis clear.

MEASLES. The prodromal presence of catarrhal symptoms, Koplik's spots, absence of pains, and character of the rashes makes the diagnosis evident.

SMALL POX In the first 3 or 4 days it is almost impossible to diagnose this disease from dengue, if the erythematous blush that often occurs on the 2nd day in small pox is present. When the true eruption of small pox comes out on the fourth day the diagnosis is clear or when on the other hand, all the acute symptoms suddenly disappear as in dengue.

DENGUE.

Occurs between 40th parallels all over the world.

Mortality nil.

Temperature rises to stadium then remission.

Two febrile attacks.

Pulse rate increases & diminishes with the temperature.

Face flushed but no icteroid tint except in rare cases on 3rd day.

Well marked rashes never petechial.

Urine normal never suppressed.

Tendency to haemorrhage trivial & rare.

YELLOW FEVER.

Only between the 40th parallels in the Atlantic.

Mortality from 15 to 85%

Temperature steadily rises.

One febrile attack.

Pulse rate diminishes as the temperature rises.

Face flushed but icteroid tint on first day.

Rare and apt to be petechial.

Urine albuminous often suppressed.

Haematemesis frequent and grave.

DENGUE.ACUTE
ARTICULAR RHEUMATISM.

Effusions into Synov-
ial cavities do not
occur.

Does occur.

Skin never flushed
over affected joints

Skin flushed.

No pain on pressure.

Very painful.

Pain not confined to
joints.

Confined to joints.

Rashes occur.

Only sudaminal & mili-
ary vesicles.

No acid sweats.

Acid sweats.

Occurs in epidemics.

Is not epidemic.

Marked depression.

Depression not marked.

Cardiac complications
unknown.

Cardiac complications
very common.

Leucopenia.

Leucocytosis.

Temperature regular.

Temperature very
irregular.

DENGUE.INFLUENZA.

Strictly limited to between 43rd parallels N. & 40th parallels S.

Distributed in all climates & all over the world.

Only occurs in hot season.

Common in cold season.

Limited to coast line & rivers.

Unlimited distribution.

Intense joint pains.

Pain not so severe.

Patient prostrated.

Ambulatory form common.

Catarrhal symptoms absent.

Catarrhal symptoms in the respiratory type of the disease.

Well marked rashes.

Rashes absent very rarely a diffuse erythema.

Complications rare.

Complications common.

Anorexia vomiting.

Gastric symptoms only in Gastro-intestinal form.

Fever of short duration.

Uncertain duration.

DENGUE.SEVEN DAY FEVER
of the Tropics.(53)
(Rogers)

PREVALENCE.

Long intervals in epidemic form attacking large proportion of residents.

Annually in sporadic form.

DISTRIBUTION

Especially attacks coast towns but spreads far inland up rivers.

Only known near the coast so far.

RACE INCIDENCE

Europeans & natives equally attacked.

Very common in Europeans rare in natives.

SEASONAL

Mostly hot months but may prevail on into cold season.

Prevails in hot and rainy seasons only.

RELAPSES

Have occurred in same year?

Very rare in same year as first attack.

PAINS

Very severe.

Not so severe.

JOINT SYMPTOMS

Very common and characteristic.

Absent or only present as slight pains.

CONVALESCENCE

May be tedious with persistent joint pains.

Rapid no after joint pains.

DENGUE.

Lasts 2 or 3 days
falling to normal
with crises or by lysis
with occasional very
short secondary rise.
Is remittent in char-
acter.

FEVERSEVEN DAY FEVER.

5-8 or more days with
typical saddle back re-
mission to 100° or 99° F.
otherwise continuous
in type.

PULSE

Varies with temperature.

Slow especially in ter-
minal rise.

PROGNOSIS is excellent as very few cases have ever been recorded of death primarily due to the disease. However in chronic invalids and aged people it may be the determining factor which breaks down the resisting power and so leads to death. This is well shown in the collection of ninety-three deaths made by the Queensland Branch of the British Medical Association in 1905.

PROPHYLAXIS. There is no doubt that in the very great majority of Towns and Cities where epidemics have occurred there should ^{never} be a return of an epidemic. The Americans in their Tropical possessions have shown how completely Yellow fever and Malaria can be stamped out; by rigid enforcement of laws dealing with standing water. Unfortunately we as yet, have not been able to educate our various populations and races up to their standard. The City fathers are still very grudging in supplying the money necessary for stamping out the annoying mosquito pest; which unless thoroughly and systematically done is of course a failure. The money spent being then regretted and the purse strings shut for many years. In certain towns enthusiasts bring their methods into disrepute. I had such an example in Delhi. A Colonel on the staff was appointed from Peshawur to command the troops in Delhi. He sent for me and complained about the mosquitoes, the terri-

-ble amount of severe fever present amongst the troops; and said if I would help him, he would clear the Cantonments of mosquitoes in six weeks and told me how he had done it in Peshawur. I observed that for a year there was not to my knowledge a pool in Cantonments which I could get at, that was not kept free from mosquito-breeding. I pointed out that the Jumna varying from $\frac{1}{2}$ to 1 mile in breadth flowed the whole length of Cantonments at the feet of the walls. That the whole surrounding country with the exception of the ridge was irrigated by canals which we could not touch. However I would loyally help in every possible way. Several small tanks were drained and emptied at much expense. Crude petroleum flowed like water. Several wells were filled up and after six weeks unceasing work on the part of the whole Cantonment staff augmented by a gang of coolies the Colonel remarked he thought he only encouraged the mosquitoes. Thus in these places where the breeding places cannot be abolished the sick must be isolated by mosquito netting properly arranged. In this way fresh infection is abolished and the wide spread dissemination prevented.

Quinine is useless as a prophylactic and no drug is known that has any influence for that purpose.

TREATMENT. There is no specific drug that will cut short the course of the fever. The only thing that can be done is to alleviate symptoms and to control the temperature. When the fever first made its appearance bleeding, emetics and drastic purgations were the usual remedies at the beginning of the attack. Even then some practitioners disagreed with these remedies and contended that they brought their patients through an attack just as quickly and far more comfortably than those who used the more heroic measures. After these remedies they used Opium and Antimonial preparations with hot baths and fomentations. To relieve the headache blisters and mustard cataplasms to the temples.

A patient who is attacked should at once be put to bed under mosquito curtains and protected from the glare of the sun. In the rare cases in which the patient has recently had a full meal and complains of nausea and cannot vomit an emetic of two drams of Vinum Antimoniale will be found to be followed by great relief. In all cases a mild purgative should be given; the best in my opinion is Calomel in doses of $\frac{1}{2}$ a grain every $\frac{1}{2}$ hour until 3 grains have been taken. If by that time the bowels have not moved two drams of magnesium sulphate or a Siedlitz powder should be given. A very light liquid diet of milk and soda water

or barley water is all that is necessary. For the pain in the head and limbs a pill of Phenacetin grains 5 Caffein gr. 1 and Codeine gr. $\frac{1}{2}$ taken three times a day will generally be most beneficial. For the joint pains hot fomentations are most comforting. 10 grains of Dever's powder at night will usually cause a good night to be passed. In some cases the pains will be so acute that a hypodermic of $\frac{1}{4}$ to $\frac{1}{2}$ of a grain of morphia will be required. If nervous symptoms supervene the bromides should be given in twenty grain doses.

For the high temperature nothing equals cold sponging with vinegar and water which usually is quite sufficient. Sodium salicylate in ten grain doses every four hours certainly keeps a check on the temperature and in some cases relieves the pain in the joints. As a rule though nothing but opium has this effect when the pains are really severe.

During convalescence a tonic such as Easton's or Fellow's syrup is of undoubted benefit. If the pains in the joints continue they should be well rubbed with A.B.C. linament and a mixture containing Potassium iodide and Sodium salicylate should be prescribed.

CONCLUSIONS.

1. Dengue is a Tropical and Sub-tropical fever limited to between the 40th parallels North & South of the Equator.
2. It occurs in the hottest and dampest seasons of the year, and the epidemic is brought to a close by the onset of the cold weather.
3. The fever is a coast disease and spreads rapidly along estuaries and rivers, and avoids altitudes.
4. Is endemic in Egypt and Syria.
5. Attacks every race, age and sex equally.
6. Is pandemic in its outbreaks, which occur at varying intervals and according to no known law.
7. The disease is due to some ultra-microscopic organism which is present in the peripheral blood.
8. The infection is carried by the *Culex fatigans*.
9. The incubation period varies from twenty-four hours to seven days but is generally between two and three days.
10. One attack usually but not always renders an individual immune for that epidemic.
11. Some individuals are naturally immune.
12. The attack is sudden in its onset and prodromal symptoms most unusual.
13. There are usually two distinct rises of temperature, but there may only be one.

14. There are usually two distinct rashes, there may be only one and in rare cases none at all.
15. No Anaemia is present. A distinct leucopenia occurs with a decrease in the number of polynuclear cells, and increase of the large mononuclears and a great increase in the number of lymphocytes.
16. The symptoms manifested vary in detail between different epidemics, and may do so in the course of the same epidemic.
17. Complications and sequelae are rare.
18. It is practically non-fatal.
19. In the four fatal cases reported intense congestion of the brain and lungs was found at the post-mortem examinations.
20. That in the beginning of an epidemic where small pox is prevalent the differential diagnosis between the two diseases for the first three or four days is extremely difficult or impossible.
21. By protection of the sick from the bites of mosquitoes the disease can be strictly limited.
22. In districts which are cleared of mosquitoes the disease is prevented from occurring although raging in the surrounding country.
23. No drug is known as a specific in the treatment of the disease or will act as a prophylactic.

APPENDIX I.

Bryden's Annual returns of the European Army of India
1871 to 1876.

The number of Dengue Cases returned with their monthly
prevalence in 1872.

	Bengal. Proper.	Dinapore. Benares & Cawnpore. Oude	Meerut. & Rohil- cund.	Agra & Central. India	Punjaub
Strength.	7,458	6,767	5,128	4,223	14,941
No. of cases in					
MARCH	1				
APRIL	214				
MAY	418				
JUNE	273				
JULY	399	7			
AUGUST	240	413		114	
SEPT.	69	630	126	811	
OCTOBER	43	449	9	172	2
NOVEM.	14	88	17	19	3
DECEM.	-	8	5	2	4
Total num. of cases.	<u>1671</u>	<u>1595</u>	<u>157</u>	<u>1,118</u>	<u>9</u>

The above statistics show how very general the disease
was in India in 1872, the only known case where the
fever left the coast and became wide-spread.

The abrupt termination of the epidemic character on the
commencement of the cold weather is very well shown.

Appendix II.

Hongkong. Latitude $22^{\circ} 16' 20''$ N.
Longitude $114^{\circ} 8' 16''$ E.

Height of Barometer taken above sea level 18 feet.

1902.

1903.

1904.

	Mean Barometer corrected and adjusted to 32° F.	Air Temperature			Tension of vapour	Relative Humidity per cent	Rainfall inches	Months of days of rain	Mean Barometer corrected and adjusted to 32° F.	Air Temperature			Tension of vapour	Relative Humidity per cent	Rainfall inches	Months of days of rain
		Mean	Maximum	Minimum						Mean	Maximum	Minimum				
January.	30.121	65.4	73.2	57.6	40.2	59	0.50	-	30.179	-	-	55.2	38.0	70	2.14	9
February.	30.250	61.9	69.4	54.5	34.7	55	0.0	-	30.269	60.7	66.4	55.1	37.9	70	0.14	2
March.	29.998	69.6	75.0	64.2	57.8	75	0.69	8	30.032	68.3	73.9	62.8	58.8	83	4.78	11
April.	29.971	75.8	81.2	70.5	67.1	71	2.86	4	30.010	74.3	79.4	69.2	64.6	76	5.71	8
May.	29.811	81.0	86.0	76.0	85.7	79	44.12	20	29.858	77.2	81.9	72.6	77.1	81	12.31	15
June.	29.716	81.3	85.3	77.4	86.8	79	21.35	13	29.823	83.9	89.5	78.3	89.1	75	23.47	14
July.	29.716	83.1	88.5	78.8	89.0	74	24.13	15	29.743	84.4	89.9	78.9	90.4	73	12.80	20
August.	29.588	84.0	90.3	77.7	89.4	71	42.0	14	29.792	83.1	88.4	77.9	87.2	75	10.77	17
September.	29.869	83.0	89.9	76.1	70.3	55	2.21	3	29.889	80.9	86.0	75.7	80.1	75	20.3	14
October.	30.029	79.4	86.2	73.6	66.0	61	1.20	3	29.904	78.7	84.6	72.9	66.5	64	1.56	7
November.	30.070	74.5	81.3	67.7	58.3	88	5.86	8	30.102	69.2	76.5	61.9	45.3	57	0.94	3
December.	30.077	72.5	83.2	61.9	47.5	72	4.43	12	30.146	62.7	70.4	55.0	35.4	55	0.0	0
Average for year.	29.935	75.9	82.4	69.9	66.0	68	14.935	104	29.979	70.9	-	68.0	64.1	71	94.65	120

267 cases of Dengue were returned by
the Bureau. All the cases occurring
in August, September and October.

Only three cases of Dengue in August.

26 cases of Dengue occurred.

APPENDIX III

CAIRO

YEARS	ANNUAL TEMPERATURE		HUMIDITY	
	1884-1886	1887	1884-1886	1887
January	53.6°F	53.8°F	70.0	72.8
February	56.6°	55.6°	66.6	71.9
March	61.7°	61.6°	57.6	63.2
April	70.2°	71.4°	46.6	49.5
May	75.2°	75.4°	48.3	47.5
June	83.3°	80.8°	42.6	47.0
July	82.4°	83.2°	49.2	52.7
August	82.0°	82.2°	55.2	59.1
September	76.8°	79.2°	62.5	63.0
October	72.4°	78.6°	62.5	69.7
November	64.2°	67.4°	67.7	69.7
December	<u>58.4°</u>	<u>58.8°</u>	<u>70.8</u>	<u>66.3</u>
Average	<u>69.6°</u>	<u>70.6°</u>	<u>58.6</u>	<u>61.0</u>

Dengue first appeared at the end of August and disappeared in December when the cold weather began.

The minimum temperature falls from 50° F. in November to 41° F. in December. From the above statistics it is evident that the disease requires the combination of humidity and heat. This occurs at the time of year when the Nile is in flood.

APPENDIX VI.

(54)

Culex fatigans.

THORAX, brown with two distinct dark lines on the denuded surface covered with pale golden curved scales and with two more or less distinct bare parallel dark lines and three rows of dark bristles.

ABDOMEN, dark brown to black with basal white or pale creamy curved bands and white lateral spots. Venter white or yellow scaled.

LEGS, dark brown bases of the femora and coxae pale, knee spot and sometimes the apex of the tibia with a faint yellow spot. Ungues of female equal and simple in male the fore and mid-ungues are unequal and uniserated, the hind equal and simple.

WINGS with the first submarginal cell longer and narrower than the second posterior cell, its stem variable in length never less than a quarter the length of the cell; posterior cross vein distant twice its own length from the mid cross vein.

FEMALE. HEAD, brown covered with pale golden brown to creamy curved scales and a few scattered black, dark brown and occasionally ochraceous upright forked scales, flat creamy white scales laterally and a faint pale narrow border round the eyes, numerous black and brown bristles; antennae dark brown with pale pubescence basal joint pale ferruginous to ochraceous, basal half of the second joint ferruginous; palpi densely covered

with deep brown scales and in some specimens with a few pale grey ones and with numerous small black bristles; proboscis covered with dark brown to violet black scales sometimes paler in the middle and with a pale apex.

THORAX, brown with two darkened lines on the denuded surface sometimes with traces of a third median line, covered with bright golden to pale golden or almost fawn coloured curved scales, some being rather broader than others, there are also three rows of black bristles, the median one ending at the bare space before the scutellum; scutellum dull ochraceous with pale golden to creamy curved scales and brown border bristles which vary in number on the median lobe, metanotum pale chestnut brown in some specimens with darker markings; pleurae pale testaceous with three or more small patches of pale almost white scales.

ABDOMEN, covered with brown to deep purplish brown or almost black scales each segment with a basal white band rather expanded in the middle and with lateral patches which shew especially in gerged specimens, posterior borders of the segments with pale golden brown hairs; venter with numerous dull scales, the first abdominal segment is dull ochraceous brown (black in gerged specimens) with two patches of dull violet black or ochraceous scales and numerous golden brown hairs.

LEGS, with the coxae deep ochraceous; femora deep brown above and at the apex, grey beneath tipped with a few yellow scales, tibia deep brown with a deep ochraceous apical spot and with pale dull brown bristles, metatarsi and tarsi deep brown to almost black with dull ochraceous reflexions in some specimens, hind metatarsi a little shorter than the hind tarsi, unguis equal simple and of moderate size.

WINGS, longer than the abdomen, the veins clothed with brown and purplish brown scales, long scales on the whole of the second and fourth long veins, also on the third, the upper branch of the fifth, and the end of the sixth, as well as on both the branches of the fork cells, the first submarginal cell longer and slightly narrower than the second posterior cell its stem short always more than one fourth the length of the cell, the stem of the second posterior cell longer than that of the sub-marginal cell but nearly as long as its fork, the posterior cross vein a little longer than the mid cross vein distant from the latter always more than its own length usually about twice its own length distant.

Halteres ochraceous, slightly fuscous at the top.

Length 4.5 to 5.5 mm.

MALE. HEAD, ornamented as in the female, antennae deep amber brown with pale bands and ^{deep} ~~app~~ brown verticillate hairs, basal joint large pale ochraceous, palpi deep ochraceous covered with brown scales, with a single pale band towards the basal third, longer than the prebescis by the last joint and nearly half the penultimate joint, the last joint tapering to a point just a little longer than the penultimate joint, clothed on each side of the last two joints with moderately long but scanty dark brown hairs which also exist at the apex of the anti-penultimate joint, prebescis deep ochraceous swollen towards the apical end covered with deep purplish brown scales, apex testaceous sharply acuminate.

ABDOMEN, narrow covered with brown scales deep purplish brown to almost black, in some specimens with basal white bands which on the last few segments spread out laterally; venter with grey or dull white scales clothed above and laterally with numerous golden brown hairs.

WINGS, with the bases of the fork cells nearly level, the first sub-marginal cell from one and a half to two and a half times as long as the cell, posterior cross vein about twice its own length distant from the mid cross vein.

LEGS, rather more ochraceous than in the female, fore and mid. unguis unequal, dark brown to black; in the fore legs both are toothed the larger one having a long blunt tooth about the middle, the smaller a sharp pointed one towards the base, in the mid legs, the unguis are very similar, but the larger tooth is a little more curved, hind unguis equal, small moderately curved.

Length 4 to 5 mm.

SUB SPECIES.

A. Type. Abdomen dusky black with basal pure white bands and basal white lateral spots; pleurae and metanotum chestnut brown; thorax with two dark parallel lines.

B. Sub species luteocinnulatus.

Abdomen dusky black or brown with basal flaxen curved bands and pure white lateral spots, pleurae and metanotum chestnut brown, thorax with traces of two parallel bare median lines.

C. Sub species Macleayi.

Abdomen brown with basal pale flaxen to almost white bands and white lateral spots, thorax with two very clear median parallel bare lines in front widening out towards the fore end.

D. Sub species Skusii.

Abdomen with pale flaxen bands and white spots thorax with traces of parallel bare lines.

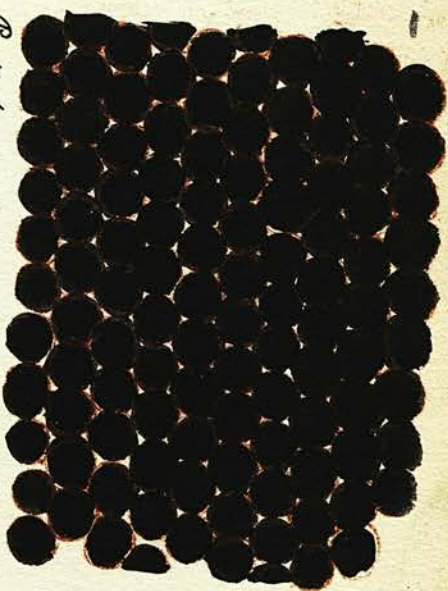
E. Subspecies trilineatus.

In which the median line of dark thoracic bristles shows as a third median line on the thorax.

The *Culex fatigans* is found in all parts of the world except temperate and Arctic regions. It is essentially a household mosquito and found wherever man goes. It can undoubtedly be spread by means of steamships and railway trains in which it is frequently found.

The larvae live in butts, tanks and other small artificial collections of water found round houses. It is especially a night mosquito and one of the most pertinacious and troublesome.

Ross calls it the grey mosquito and found it to be the carrier of the *Proteosoma* of birds. Manson reports it as being the carrier of *Filaria Sanguinis Hominis* and now must be added the organism of Dengue fever.



Part of a raft of eggs seen perpendicularly from above. The regular and geometrical arrangement of the eggs is well shown.



Eight rafts of the eggs of *Culex fatigans* (slight magnification). The four upper ones beginning to hatch out.



A group of separated eggs as seen under a low power of the microscope.

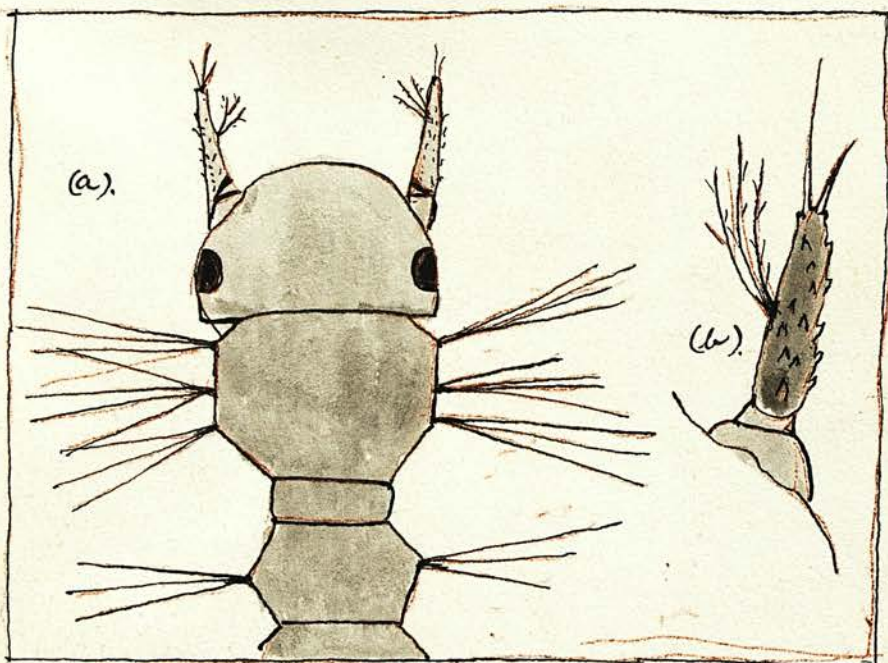


Rafts and fragments of rafts of the eggs of the *Culex fatigans*. (very slight magnification).

B



The almost adult larva of *Culex fatigans* showing the long respiratory tube.



a. Semi diagrammatic design of the head and first two segments of the larva of *Culex fatigans*.
b. Enlarged antenna of larva.



Lateral view of the pupa of *Culex fatigans*.

2

Culex fatigans.

Adult Male.



Adult Female.



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